

DIAGNOSIS OF PULMONARY REGURGITATION BY A DYE METHOD

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Graham Steell (1888) first described a variable and at times indistinct diastolic murmur over and slightly below the pulmonary area: this he attributed to "pulmonary regurgitation . . . occurring independently of disease or deformity of the valves, and as the result of long-continued excess of blood pressure in the pulmonary artery." He later (1895) reported 60 cases of mitral stenosis and considered that only one had the murmur of "high pressure" in the pulmonary artery. Kezdi *et al.* (1955) reviewed 95 cases of mitral stenosis and found only one that they considered had the functional murmur of pulmonary regurgitation. This incidence of pulmonary regurgitation is low and reflects the strict criteria that were applied in order to arrive at a certain diagnosis.

In this department consideration of the diagnosis of pulmonary regurgitation seemed to arise more frequently, and there was often disagreement among observers about patients with early basal diastolic murmurs. This arose partly because some of these murmurs were soft and transient and partly because there was often little supporting evidence to distinguish pulmonary from aortic regurgitation. The question particularly came up in patients with rheumatic heart disease. For these reasons an attempt was made to demonstrate pulmonary regurgitation by a dye method in 9 patients undergoing cardiac catheterization.

MATERIAL AND METHOD

The nine patients investigated fell into three groups: Group A, three patients who were catheterized for routine purposes and were not thought to have a Graham Steell murmur; Group B, four patients in whom the presence of a Graham Steell murmur was debatable; and Group C, two patients in whom a Graham Steell murmur was clinically agreed to be present. The diagnosis of a Graham Steell murmur was made clinically on the basis of a blowing early diastolic murmur heard in the left 2nd and 3rd intercostal space in the absence of peripheral signs of aortic regurgitation and in the presence of a strong systolic pulsation over the area of the right ventricle, a palpable pulmonary artery impulse, an accentuated second sound in the pulmonary area, X-ray signs of pulmonary artery enlargement, and electrocardiographic evidence of right ventricular hypertrophy.

At right heart catheterization a number 8 catheter was placed 1–2 cm. distal to the pulmonary valve, and a number 9 catheter with multiple perforations at the end (Goodale-Lubin tip) was placed in the right ventricle just below the pulmonary valve. In the first three studies (patients B4, B5, and B6), 2 ml. of Evans blue (T-1824) were injected into the pulmonary artery in less than 1 second, and at the same instant fractional collection of blood from the right ventricle was begun with a Cornwall double valved syringe at a rate of 2 ml. per second. This had the disadvantage that, with a single bolus of dye rapidly injected into the pulmonary artery, streaming from the tip of the catheter was great and regurgitant dye that appeared transiently might be missed with the interrupted method of sampling. Therefore, in the remaining six patients the dye injection consisted of a mixture of 2 ml. of 1 per cent Evans blue and 8 ml. of saline which was infused into

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the pulmonary artery catheter at a rate of 1 ml. per second for ten seconds. In both methods sampling from the right ventricle continued for at least ten seconds. The samples were analysed for dye content on a Unicam spectrophotometer (SP-600). Cardiac outputs were determined by direct Fick and dye methods, and oxygen content of blood by the Haldane method.

RESULTS

The results are summarized in the table. In two patients in Group A a slight amount of dye was obtained from the catheter in the right ventricle when dye was injected into the pulmonary artery. In A1 it appeared during the second to sixth seconds after the single bolus injection of dye, and in A2 it appeared only during the sixth and seventh seconds after the beginning of a ten-second dye infusion. In one patient (C8) in Group C there was pronounced regurgitation of dye through the pulmonary valve lasting as long as did the infusion. No dye was demonstrated in the samples from the remaining patients.

TABLE
RESULTS IN SUSPECTED OR PROVEN PULMONARY REGURGITATION

Patient	Clinical impression	Pressures in mm. Hg				Cardiac output (l./min.)	Pulmonary arteriolar resistance (dynes/sec./cm. ⁵)	Diagnosis of pulmonary regurgitation	
		RA	RV	PA	PC			Clinically	By dye
A1	MS, AR, AS	16/5	42/7	42/26	24/18	3.9 (dye), 4.1 (Fick)	195	None	?
A2	MS, AS, MR	5*	28/6	28/13	27*	4.9 (dye)	161	None	?
A3	MS	5*	32/3	33/17	23*	4.4 (Fick) 4.1 (dye)	182	None	None
B4	MS, MR	15/10	32/9	38/23	15/10	2.9 (Fick)	632	?	None
B5	MS, MR	15/7	34/6	42/27	16/10	—	—	?	None
B6	MS	11/6	39/3	40/16	26/13	4.7 (dye)	259	?	None
B7	MS, MR, AS, ?AR	16/9	48/7	46/27	—	—	—	?	None
C8	Reversed ASD	—	117/8	117/72	5*	6.7 (dye)	979	Definite	Definite
C9	Reversed PDA	6*	102/9	102/72	—	3.7 (Fick)	—	Definite	None

* Mean pressure

MS=mitral stenosis.
MR=mitral regurgitation
AS=aortic stenosis.
AR=aortic regurgitation.
ASD=atrial septal defect.

PDA=patent ductus arteriosus.
RA=right atrium.
RV=right ventricle.
PA=pulmonary artery.
PC="pulmonary capillary".

Dye delivery by 10-sec. infusion except in patients B4, B5, and B6 when one rapid bolus used.

DISCUSSION

Two patients in the first group, who were thought clinically to have no Graham Steell murmurs, unexpectedly showed regurgitation of dye into the right ventricle during the infusion in the pulmonary artery, but in both instances the dye gave only a minimal reading on the spectrophotometer and was not visible to the naked eye. In one of these patients (A2), coronary or intercostal

recirculation could account for the dye in the right ventricle, since it appeared only during the sixth and seventh seconds after beginning the infusion. In the other (A1), the dye appeared too early for this explanation, and it is possible that he had a slight degree of pulmonary regurgitation not suspected because of the over-shadowing of aortic regurgitation.

Only one patient showed pronounced regurgitation of dye through the pulmonary valve: this man (C8) was thought clinically to have a Graham Steell murmur, and he was the patient with the highest pulmonary artery pressure recorded in the study. The other patient (C9) in whom the diagnosis of a Graham Steell murmur was made confidently showed no regurgitation of dye, nor did any of the patients in the questionable group. Two explanations are possible: (1) none of these patients had any regurgitation at the valve, or (2) the method as performed here is inadequate to demonstrate small amounts of regurgitation. With regard to the latter, it is conceivable that when dye is injected into the pulmonary artery a streaming effect of the dye jet may carry the dye beyond the area of regurgitating blood; this possibility might be overcome by making multiple perforations near the tip of the delivery catheter, thus ensuring better mixing in the pulmonary artery. In addition, accurate positioning of the two catheters immediately distal and proximal to the valve seems to be of paramount importance (Bajec *et al.*, 1958), and a slight error in positioning may produce false negative results.

While this method demonstrated pulmonary regurgitation in one patient, there are apparently pitfalls. This direct method is not superior to the indirect one of diagnosing an incompetent valve by recording two peripheral dye curves, one distal and one proximal to the valve (Lendrum and Shaffer, 1959).

SUMMARY

An attempt was made at cardiac catheterization to demonstrate regurgitation of dye through the pulmonary valve in a series of nine patients with basal diastolic murmurs, some of whom were thought clinically to have Graham Steell murmurs. Evans blue dye was injected into the pulmonary artery and samples of blood for dye analysis were obtained by a fractional collector from the right ventricle. The demonstration of regurgitant dye did not occur in all instances in which it was clinically suspected, and the difficulties in this method of proving pulmonary regurgitation are pointed out.

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